THE RHODE ISLAND MEDICAL JOURNAL

Owned and Published by the Rhode Island Medical Society. Issued Monthly

VOLUME XVI No. 9 | Whole No. 288 | PROVIDENCE, R. I., SEPTEMBER, 1933 | PER YEAR $2.00 SINGLE COPY 25 CENTS

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ENTERED AS SECOND-CLASS MATTER AT THE POST OFFICE AT PROVIDENCE, R. I., UNDER ACT OF MARCH 3, 1879

THE RHODE ISLAND MEDICAL JOURNAL

The Official Organ of the Rhode Island Medical Society
Issued Monthly under the direction of the Publication Committee

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ORIGINAL ARTICLES

MODERN IDEAS IN REGARD TO EPILEPSY*

By WILLIAM NEWTON HUGHES, A.M., M.D.
112 WATERMAN STREET, PROVIDENCE, R. I.

Lennox and Cobb, in their monograph on "Epilepsy, Medicine Monographs, Volume XIV," Williams & Wilkins Company, 1928, summarized very clearly most of the work on epilepsy which seemed scientifically sound at that time. I have followed their outline to a large extent in preparing this paper.

"Modern Problems in Neurology," S. A. Kinnier Wilson, William Wood and Co., 1929, gave an excellent summary of the author's study on epilepsies and narcolepsies and contributed much to our present ideas.

Talbot's "Treatment of Epilepsy," The Macmillan Co., 1930, summarized much of the important work on epilepsy up to that time.

In the May 1930 "Archives of Neurology and Psychiatry," Vol. 23, No. 5, Temple Fay summarized much of the work on dehydration in epilepsy and reported its therapeutic effect on fourteen epileptic patients over a two and one-half year period.

In the October 1931 "Archives of Neurology and Psychiatry," Vol. 26, No. 4, Weiss and Lennox showed that convulsions may occur even with the cerebral vessels dilated under the influence of histamine.

In the May 1932 "Archives of Neurology and Psychiatry," Vol. 27, No. 5, a special article on "Causes of Epilepsy" by Cobb reviewed briefly many of the known facts in regard to epilepsy.

In the December 1932 "Archives of Neurology and Psychiatry," Vol. 28, No. 6, articles on the cerebral circulation by Cobb and Finesinger and by Chorobski and Penfield established new physiological and anatomical facts which proved assumptions previously made in regard to epilepsy.

In the three books and four articles just mentioned can be found bibliographies which will cover all the references in my paper. I have the highest regard for the work done by Lennox and Cobb on epilepsy and for their evaluation of the work of others. I can recall no instance in which their ideas and mine on epilepsy are not in entire agreement.

In writing this paper, I planned to emphasize the use of dehydration in convulsive and allied conditions because I had found that the majority of physicians had no very clear ideas in regard to its value. As some knowledge of modern ideas in regard to convulsive and allied conditions is necessary for a proper understanding of the use of dehydration, an exposition of these ideas takes up much of the paper.

Epilepsy is considered by most physicians today as a convulsive state or seizure which tends to recur, a symptom rather than a disease. It is, to quote Lennox and Cobb, "a syndrome characterized by the sudden appearance of paroxysms, of which convulsive movements or loss of consciousness or both, are a principal element." This definition will include the seizures which occur in Jacksonian epilepsy, pycnolepsy, uncinate epilepsy, so-called idiopathic epilepsy, symptomatic epilepsy, and hystero-epilepsy, and both types of seizures, the grand mal and the petit mal. As allied to these convulsive states, and perhaps partially explained by similar ideas, I shall mention only migraine and narcolepsy, though there are many other conditions, of course, which might be described.

Migraine, as you know, is a severe, recurrent or periodic hemicrania with or without ophthalmic and abdominal symptoms. It has been called a sensory seizure. Narcolepsy is a state of recurrent paroxysmal diurnal sleep or apparent sleep regardless of how dangerous the situation in which it occurs may be. It is often associated with catalepsy, a state of sustained immobility with or without clouding of the sensorium, and with cataplexy, a condition with sudden transient physical powerlessness under the influence of the emotions.

The convulsive capacity, or whatever you wish to call it, for these states is apparently inherent. It is so great that convulsions may occur even with the cerebral blood vessels dilated with histamine, as shown by Weiss and Lennox (1931). As proof

*Read before the Rhode Island Medical Society, Providence, R. I., March 2, 1933.

of this inheritable trait, Lennox and Cobb (1931), in their study of 1,086 non-traumatic and non-institutionalized epileptic persons with 9,139 near relatives, found eight times as many epileptic relatives per thousand as in a normal control group of 250 persons with 1,896 near relatives. The near relatives consisted only of parents, siblings, and offspring. They also state that more epilepsy is found among the children of migrainous parents than among the children of epileptic parents. This indicates a certain group of "silent carriers" of the tendency, whom it would be difficult to sterilize or to prevent from marrying in the hope of cutting down the inheritance of the convulsive capacity. The convulsive capacity may be brought into play by a number of different precipitating factors, such as functional physical and chemical changes within the brain tissue, mostly associated with acid-base and water balance, psychogenic factors, gross and microscopic abnormalities in brain structures, and abnormalities in body structure other than the brain, e. g., in the gastro-intestinal tract, respiratory tract, endocrine glands, metabolism, sympathetic system, and intracranial circulation.

How the neurological mechanism of a convulsive state occurs is not exactly known. Numerous theories have been advanced, but no exact localization of the focus from which fits arise can be made. Some believe that the cortex is the responsible factor; others that the brain stem without the cortex is the responsible factor; while still others feel that the convulsion may start in the cortex or in the brain stem or even in the spinal cord, and it is unquestionably true that convulsions can be set off by disturbances in various parts of the central nervous system. The four chief theories which have been advanced to explain the mechanism of convulsive states are the irritative, the release, the short circuit, and the explosive.

The irritative theory is the one which is most commonly known. Irritation or stimulation of the cortex by various factors, such as scar tissue, hemorrhage, meningeal inflammation, etc., is supposed to result in convulsions. Jacksonian epilepsy seems to be partially explainable by this theory.

The release theory, perhaps better known as the theory of cortical inhibition, assumes that when inhibiting impulses from the entire cortex are suddenly and temporarily removed, the brain stem and spinal cord will function explosively in the typical epileptic seizure. The decerebrate rigidity of animals suggests this theory. The occurrence of a Babinski reaction during a petit mal attack in which the cortex could not be considered to have become exhausted is evidence of interference with cortical function and may suggest some inhibition there.

The short circuit theory is best supported by the work of Fulton, and Dr. Southard was one of its foremost advocates. According to it, associative or internunciatory neurones are supposed to delay action between afferent and efferent neurones, and there is an inhibitory block to motor action at the various motor levels in the nervous system, e. g., the spinal, cerebellar, basal, and cortical. When these associative neurones are fewer in number than normal, due to agenesis, destruction by birth trauma or head injury or some other factor, there is less delay in afferent-efferent response. A shorter path is taken, and then disorderly, convulsive, explosive phenomena result. The short circuit theory is really a release theory, but only small areas of the nervous system are inhibited, while according to the main release theory the entire cortex is inhibited.

The explosive theory is perhaps the one which most appeals to modern research workers, and it is the one with which further research is apt to be most productive. Some sudden change in nerve cells is conceived to occur which makes them react explosively over widespread areas. This change within the nerve cells is comparable to that which occurs in anaphylaxis, hyper insulinemia and states of anoxemia and alkalosis.

Probably the neurological mechanism of a seizure is not explained by any one of these theories, but various combinations of them may be needed to explain the mechanism of any seizure. At the present time the explosive theory seems to be more satisfying and stimulating than any of the others.

Of course, the important thing is not the neurological mechanism or course of events in a seizure, but why the seizure occurs. I have given brief suggestions in covering the neurological mechanism as to how a seizure occurs. I shall describe the modern theory of Lennox and Cobb as to why the seizure occurs, because I believe that their theory is the best and represents the most careful work. It is somewhat as follows: In certain people who have an inherent, possibly inherited, convulsive capacity, various factors, psychogenic, gastro-intestinal, respiratory, endocrine, metabolic, intracranial, etc.,

cause sympathetic stimulation which results in contraction of cerebral arteries and consequent cerebral anemia with decreased blood flow in the capillaries. This sympathetic stimulation and anemia may be focal and may occur in different parts of the brain. It leads to deficient oxygenation and consequently alkalosis, which results in increased permeability of the capillaries with a pouring of fluid and inorganic base from the blood into the tissues, a tissue edema, which in turn results in some increased intracranial pressure and so more anemia, etc.,—a vicious circle. In addition, the alkalosis causes a less efficient use of what little oxygen there is and so an increased tissue-anoxemia, which results in more alkalosis and tissue edema. In this way another vicious circle is started. Somehow or other this anoxemia, alkalosis, edema, change in electrolyte equilibrium, increased intracranial pressure, one or all start up an explosive action in nerve cells which results in an epileptic seizure. Then, due to apnoea and muscular contractions, lactic acid and CO_2 collect in the tissues and give a condition of acidosis which initiates a reversible reaction leading to better utilization of oxygen, less edema, a lowered intracranial pressure, a restoration of circulation with a release of arterial spasm and somehow or other a release of general muscular spasm.

I shall try to give some idea of the evidence, experimental and otherwise, upon which the different parts of this theory are based. The inherent convulsive capacity or unknown constitutional element is assumed because only a small minority of people who present the various precipitating factors of seizures actually have seizures. Proof that this convulsive capacity may be inherited in certain cases has already been given.

Anatomy and physiology show that the sympathetic autonomic system is richly distributed to the gastro-intestinal tract, respiratory tract, endocrine glands, and blood vessels, and that it has much to do with their proper function. Clinical experience indicates that at certain times the cranial-sacral autonomic seems to predominate in function and that at other times the cervical-thoracic sympathetic seems to predominate in function. Various physiological or anatomical changes in the gastro-intestinal tract, respiratory tract, endocrine glands, and blood vessels such as constipation, intestinal ptosis, body posture, fatigue, deficient pituitary secretion, increased adrenalin secretion, etc., might result in sympathetic stimulation or cranial sacral autonomic fatigue with diminished function and so an increased sympathetic function through lack of inhibition.

The association of emotions with the sympathetic nervous system is too well known to need further comment.

Stohr (1922) gave convincing illustrations of nerves and nerve endings on the fine pial blood vessels. Penfield (1932) described nerve fibers on intracerebral blood vessels. Forbes and Wolff (1928) showed through a window in the skull contraction of the pial arterioles on stimulation of the cervical sympathetics and dilatation of these vessels on vagus stimulation. Stimulation of the cervical sympathetics on one side of the neck resulted in contraction of the pial arterioles on the same side of the brain, while stimulation of the vagus on one side resulted in dilatation of pial vessels on both sides of the brain. Cobb and Finesinger (1932) showed that vasodilator fibers pass up the vagus trunk to the medulla oblongata, then to both facial nuclei through an intramedullary route yet unknown and out along the facial nerves as far as the geniculate ganglion. These fibers were traced by Chorobski and Penfield (1932) without interruption at the geniculate ganglion into the greater superficial petrosal nerve to connect with the plexus of nerve fibers on the internal carotid artery. The vasodilator fibers then seem to spread along the large cerebral arteries and their branches just as do the sympathetic nerves which join the carotid artery at a lower level.

Spielmeyer (1924-1927) found in Ammon's horn and the cerebellum in 80% of 126 necropsies on epileptics small focal destructive lesions which presented the picture of an ischemic destruction—cell degeneration, lipoid products of degeneration and gliosis similar to that found in arteriosclerosis. He believes that these changes are caused by a functional circulation change—a vasoconstriction.

Red blood cells with oxygen bound to their hemoglobin are much less numerous in the areas of local anemia which results from contracted blood vessels. Deficient oxygenation then results, as there is much less oxygen available for tissue uses than is usually the case. Acute anoxemia is accompanied by alkalosis due to the increased lung ventilation and elimination of CO_2 which occurs during the anoxemia. As a result of the "blowing off" of CO_2 with its tendency to increase blood alkalinity, base

substances leave the blood and enter the tissues in order to keep the hydrogen ion concentration of the blood fairly constant. Acute alkalosis results in edema and, presumably, increased permeability of tissues is a corollary of this edema. Edema in the brain tissue, which is enclosed by the meninges and the skull, produces increased intracranial pressure.

Jacobi (1926) and Forbes and Wolff (1928) showed contraction of pial arteries with overventilation and alkalosis. It is evident that alkalosis increases anoxemia through contraction of arterioles with a diminution in red blood cells with their oxygen bound to hemoglobin.

In alkalosis, also, the blood gives up oxygen less readily because the alkalosis has raised the oxygen dissociation curve. This decreases still further the utilization of oxygen by the tissues.

The following physiological changes in the brain, as enumerated by Lennox and Cobb, have been shown to prevent seizures and their opposites to precipitate them:

(1) A rich oxygen supply.

(2) Acidosis produced by fasting, by a ketogenic diet, by acids or acid-forming salts, and by inhalation of CO_2.

(3) A low chloride and a high calcium content.

(4) Dehydration of tissues.

(5) Decreased permeability of tissues.

(6) Decreased intracranial pressure.

(7) An unimpaired intracranial circulation.

I shall briefly discuss some of the evidence connecting the above physiological changes in the brain with seizures. Lennox (1928) demonstrated that in certain patients who had frequent petit mal seizures, attacks could be precipitated by decreasing the oxygen content of the respired air by rebreathing air from which the CO_2 was absorbed and that attacks could be prevented by increasing the oxygen content of the respired air.

Lennox and Cobb (1928) fasted 27 adolescent and adult patients of the non-institutional type and obtained definite improvement in about one-half of the cases during the fast. They believe that the most probable explanation of the improvement during the fast is the accompanying acidosis or closely related physico-chemical changes in the nerve cells.

Helmholz (1927) at the Mayo Clinic reports that of 91 children, adequately treated by a ketogenic diet, 31% have been free of attacks and an additional 23% have been definitely improved.

Lennox (1927), following the ingestion of NH_4Cl, $CaCl_2$ and NH_4NO_3, obtained marked temporary reduction in the frequency of seizures, though in a few instances seizures returned in seven to ten days in spite of the continued administration of these salts. Lennox and Cobb (1928) showed that seizures cease on rebreathing air from which the CO_2 has not been removed.

The return of seizures in spite of the continuation of acid salts suggests that, even though acidosis still persists, its beneficial effect is over-balanced by an increased concentration of chlorides in the tissues. The feeding of NH_4NO_3 tends to produce an acidosis due to increased blood chlorides.

Convulsions in tetany are increased by any salt which will precipitate calcium in the tissues and body fluids.

That excessive intake of fluid predisposes animals to convulsive seizures is well shown by the "water intoxication" experiments of Rowntree (1926) in which dogs given excessive fluid by stomach tube developed typical convulsions and often died in status in four or five hours.

Weed (1921) noted convulsive seizures in animals by giving excessive fluid and also he found that the seizures had a more rapid onset if hypotonic solutions were given by vein.

Elsberg and Pike (1926) pointed out in animals that when hypotonic solutions had been given intravenously or when intracranial pressure had been increased, convulsive seizures could be produced by approximately one-half the dose of absinthe usually required to produce seizures in normal animals and that if the animals were dehydrated by the use of hypertonic solutions, twice the usual convulsant dose of the drug was required to cause an attack.

Drabkin and Ravdin (1927) found that in dehydrated animals convulsions did not occur after the same dose of insulin which caused convulsions in the normal controls.

Temple Fay (1930) reported 14 patients with convulsive seizures treated by dehydration for two and one-half years, and eight treated for less than one year. He found that proper control of fluid intake, combined with dehydration, greatly diminished the tendency to convulsive seizures in certain cases representing various types of epilepsy.

The cessation of seizures which may occur after lumbar puncture is well known, and its effect is probably due to relief of intracranial pressure and brain edema—a pressure and a dehydrating effect.

In susceptible individuals, seizures are often produced by the subcutaneous administration of pituitary extract which results in water retention and brain tissue edema. Occasionally seizures which are chiefly hysterical in origin are diagnosed by this means, as they do not seem to occur with this increase in water retention as easily as do other types of seizures.

The following diseased conditions in which convulsions often occur are associated with brain edema: eclampsia, alcoholism, cerebral trauma, birth injuries, apoplexy, acute meningism, etc.

That retention of fluid in the tissues is a factor in producing seizures is also shown by the following facts:

(1) Alkalosis which gives water retention tends to produce seizures.

(2) The drinking of large quantities of water, if associated with water retention in the body, tends to produce seizures.

(3) Acute or chronic infections which retain water in the body, perhaps to a certain extent through general vasoconstriction, tend to produce seizures.

(4) Increased concentration of chlorides in the tissues with resulting water retention tends to produce seizures.

(5) Acidosis, which gives water loss from the body, tends to prevent seizures.

(6) Restriction of fluid intake tends to prevent seizures.

(7) A low salt diet, which tends to prevent water retention in the body, tends to prevent seizures.

(8) Bromide medication, which tends to push chlorides from the body and in this way to give water loss, tends to prevent seizures.

There are comparatively few statistics which show the value of restriction of fluids alone. Temple Fay reports a small number of epileptic patients, as I have previously noted, in which he shows diminution in the number and frequency of seizures by drastic water restriction. Talbot believes that water restriction works best in adult epilepsy. The ketogenic diet which results in acidosis and loss of water from the tissues shows in children about 31% cessation of seizures and an additional 23% improvement.

Dehydration therapy is being used by many of the best neurological clinics in the country as an aid in the treatment of convulsive states, and I hope that I have been able to set forth enough facts to make fluid restriction and other dehydration therapy seem at least theoretically rational in the treatment of convulsive states. Many physicians at the present time are forcing fluids in these conditions. I have had one patient whose family doctor advised him to drink a great deal of sodium bicarbonate water to improve a transient skin condition, even though the patient told him that he had been advised to restrict his fluids because of epilepsy. The patient had the only seizure which he had had in over a year within a few hours after he had taken the first two glasses of this sodium bicarbonate water. Both the water and the sodium bicarbonate were not advisable, as has been shown in this paper. Personally, I am convinced that dehydration procedures are of great aid in controlling convulsive and allied conditions, and actually the real reason for this paper was to set forth some of the theoretical reasons for restricting fluids in convulsive states.

Syz (1927), using acid fuchsin as a convulsant in frogs, showed that injury to any part of the central nervous system caused an increase in the permeability of the whole brain and cord to this dye with a resulting increase in susceptibility to convulsions.

The experiments of Elsberg and Pike with intravenous hypotonic and hypertonic solutions as mentioned previously show that increased intracranial pressure is a factor in producing convulsions and that decreased intracranial pressure is a factor in preventing them. Clinically many conditions with increased intracranial pressure are associated with convulsive seizures in many cases, e. g., eclampsia, uremia, alcoholism, head trauma with brain injury, acute meningism, etc.

Spielmeyer's necropsy reports previously mentioned give anatomical or histopathological evidence of an impaired intracranial circulation in epileptics.

Stewart, Guthrie, Burns, and Pike (1926) found that if the intracranial circulation were interrupted for a period of minutes, the animal used later devel-

oped muscle twitchings and convulsions. Substances which cause constriction of cerebral arteries seem to tend to cause convulsions in those who are susceptible. Among chemicals commonly used experimentally for this effect, caffeine, ergot, nicotine, and camphor may be cited. Luminal which dilates cerebral vessels tends to prevent seizures. The convulsions which may occur in paroxysmal tachycardia are regarded as due to impaired intracranial circulation, or anemia. (Barnes, Mayo Clinic, 1926.)

Encephalograms and ventriculograms in convulsive states at times show evidence of interference with cerebro-spinal fluid circulation. Encephalograms in addition show an increase of fluid in the subarachnoid pathways and often cyst formation. According to Temple Fay, the spinal fluid is absorbed chiefly through the Pacchionian granules. In epilepsy he has shown that these granules are often diseased, and it is assumed that the circulation of fluid in the cerebro-spinal pathways is diminished in many cases.

According to the ideas mentioned in this paper, I shall attempt to rationalize for you a part of the treatment of patients with convulsive states and with the allied conditions, migraine and narcolepsy. First, of course, various diagnostic procedures are utilized to find out which physiological changes in the brain seem most important in the individual cases that are being studied. An attempt is made to explain to the patients the therapy advised and to connect it with the physiological factors which seem to influence their seizures. To maintain relative dehydration I suggest that they take not over one teaspoon of any liquid at one time and that they shake no salt on their food. I also ask them to restrict carbohydrate foods which give water retention in the body, to restrict to a lesser extent protein foods, and to force fat foods which tend to give water loss through a tendency to produce acidosis. In certain cases NH_4Cl or $CaCl_2$ are given temporarily to aid in pushing fluids from the body through their tendency to produce inorganic acidosis, and they are stopped in four or five days so that the chloride concentration in the tissues will not become too high. The ketogenic diet seems to work best in children and it is used at times to produce acidosis and dehydration. Occasionally magnesium sulphate is given because it tends to reduce intracranial edema and intracranial pressure and to produce acidosis. Also, it takes care of any constipation that might occur and result in sympathetic stimulation with cerebral vasoconstriction. At times calcium is given in the hope of increasing the calcium content of the tissues and so controlling the seizures. In many cases bromides in addition are given to force chlorides and with them water from the body. In many other cases luminal is given for dilatation of constricted cerebral vessels. Alkalis, e. g., sodium bicarbonate, which tend to produce alkalosis are forbidden. Alcohol which tends to give water retention is forbidden. To aid in getting a good oxygen supply to the brain I advise daily exercises of such a character as to improve the abdominal muscles chiefly and to promote more lung ventilation. Adequate rest and relaxation, especially after meals, is urged in the hope that it will tend to combat angiospasm of cerebral vessels. And last, but by no means least, the general physical and mental hygiene is given very careful attention in the hope of combating infections and emotional conditions which might give cerebral angiospasm.

By maintaining some such regime as described for a long period of time, it may be possible to produce some permanent change in nerve tissue which will tend to prevent a recurrence of convulsions. The fact that convulsions do not occur immediately if the ketogenic diet is stopped tends to give some support for this belief.

Summary

Evidence has been presented to show that the occurrence of epileptic seizures is markedly influenced by the following physiological conditions—an inherent convulsive capacity which is undoubtedly inherited to a certain extent in many cases; hydration in brain tissue; increased permeability of capillaries in brain tissue; change in electrolyte equilibrium in brain tissue; increased concentration of chlorides in brain tissue; decreased concentration of calcium in brain tissue; vasoconstriction of cerebral vessels; gross or focal cerebral anemia; impaired intracranial circulation of blood or spinal fluid; increased intracranial pressure; balance between cervical-thoracic and cranial-sacral autonomic systems; direct cortical "irritation"; brain tissue defects of an embryological or acquired nature; alkalosis; and anoxemia.

Physiological methods of treatment for epilepsy are being used extensively, and I believe that they are of much value.

CONGENITAL DISLOCATION OF THE HIP*

By JOHN RIDLON, M.D.
NEWPORT, R. I.

When the head of the femur is found dislocated from its socket, and there having been no known injury that could have caused it, it is called a congenital dislocation. Spontaneous dislocation would be a better name, because the dislocation is not always present at birth. It may take place later on. But the defect in the joint that makes the dislocation possible is really a congenital defect.

The real congenital defect is in the acetabulum. It is shallow — saucer-shaped, instead of cup-shaped. Sockets have been found pear-shaped at open operations, with the narrowed part uppermost. This may be due to the pressure from the femoral head as it gradually passed upwards. It is believed that there are congenital dislocations while the child is in the uterus, because such seem to have been demonstrated; and it is believed that some dislocations happen at birth, but this has not been proven. Rarely are congenital dislocations discovered until after the child has walked; but I have seen two cases where the mother noticed a short leg and brought the child for examination, and I found the hips dislocated. But in both of these cases when the children were returned for operation between two and a half and three years of age, the hips were no longer dislocated. Around the border of this shallow socket is the cotyloid ring, and this is always more or less narrowed, perhaps from the pull of the capsule. The capsule is elongated, and if the displacement is considerable it may be narrowed—hour-glassed—in its middle part; and its lower portion may be drawn across the lower portion of the socket forming a kind of pocket, which has been likened to a hymen. The dislocated head is not normally rounded, perhaps because it has not been in a normal socket. It may be shaped somewhat like the end of a lemon, and in older children it may be of a mushroom shape. The ligamentum teres is elongated, and sometimes is also enlarged. All these changes obstruct a manipulative replacement of the head. Rarely is the operator conscious of the feeling of contact of the cartilage of the head with the cartilage of the socket. The best replacement by manipulation is usually to have the head so placed, and held, that it looks through the narrowed capsule and constricted cotyloid ring into the shallow socket more or less filled up with the round ligament. But to replace these hips by an open operation also has its handicaps. For if the capsule and hymen are slit, they cannot be effectively sutured, and that leaves a weakened support. The round ligament cannot be otherwise disposed of except by removal, and that removes necessary blood-supply from the head which already lacks normal X-Ray density. The socket cannot be deepened without leaving a stiff joint—provided the hip stays in the socket. A congenital defect is a congenital defect, and can never be made quite like a normal condition.

Now these shallow sockets vary in degree. Some can never by any means that we now know of be made to retain the replaced femoral head and give a useful range of motion. Then they range all the way up to the defective socket that grasps only half of the femoral head but retains it unless subjected to a traumatism.

In 1915 I reported the case of a man, a policeman, 54 years old, who had never had any subjective hip symptoms—who thought his hips perfectly normal, whose one femoral head was in contact with the acetabulum for only three-fifths of its articular surface and in the other hip for only half its articular surface. I have seen a hip spontaneously dislocate in a child of three years, and I have seen a hip slip out in a boy of 14 years from simply bending backwards in a gymnasium. It is my opinion that all hips that dislocate from traumatism are more or less defective—have more or less shallow sockets; and that a normal hip, when subjected to a sufficient traumatism, will suffer a fracture of the femoral neck before a dislocation will occur, or a breaking off of a part of the rim of the socket will take place.

There are no subjective symptoms from a congenitally dislocated hip until late in life, and sometimes not then. I have a friend, a doctor, now past seventy years, with two dislocated hips. He can still play 18 holes of golf without fatigue, and says that it is not necessary to operate on congenitally dislocated hips—that they are not a handicap. The objective symptoms are a limp from one short leg, or a waddle from two short legs, lordosis of the lumbar spine, prominent buttocks, flexed thighs, and adduction of the thighs. When both hips are dislocated, the perineum is broadened from side to side. In a child that has not walked, the upward displacement of the femoral head may be only from one-fourth to one-half inch, while in a child of fourteen years it may amount to more than

*Read before the Rhode Island Medical Society, December 1, 1932.

three inches of real upward displacement, and the practical shortening from the flexion and adduction may amount to five inches. But there are other things that give just this picture, namely, a congenital absence of the femoral heads and necks; and coxa vara.

The certain diagnosis should be made by the fingers of the examiner. A dislocated femoral head can be felt by the fingers of the examiner. If he cannot feel it, he is not competent to replace it. If the head is dislocated backwards and upwards, it can be felt under the buttock muscles, and inward rotation of the thigh makes it more easily felt, but it cannot be felt in front. If the head is dislocated upwards it can be felt at the back when the thigh is rotated inwards, and at the front when it is rotated outwards. But if it is dislocated forwards and upwards it can be felt somewhere near the anterior iliac spine, and more distinctly when the thigh is rotated outwards, but it cannot be felt at the back when the thigh is rotated inwards. An X-Ray film may be a comfort in confirming the diagnosis, but like many laboratory findings must not have precedence over the clinical evidences. When the picture presented by the patient—I mean the posture when the patient stands—is typical of a congenital dislocation, unilateral or bilateral, and the examiner cannot find the femoral head with his fingers, and begins to doubt the accuracy of his sense of touch, an X-Ray film showing a coxa vara or a congenital absence of the femoral head and neck restores one's morale.

If these dislocations are discovered at or soon after birth the upward displacement of the femoral head may be only half an inch, or even less, but they are definitely out of the socket—not subluxations, if by subluxations one means a partial displacement. After a child has walked for a year the upward displacement may be an inch or more. By ten years the displacement may be two inches or more; and by fifteen years there may be a practical shortening of five inches. Most femoral heads are displaced more or less directly upwards—supracotyloid dislocations. Some years ago I thought that most were backwards and upwards. Now I believe that these displaced backwards and upwards rank next to the supracotyloid; and a few are displaced forwards and upwards. I was the first surgeon to report a case of forward and upward dislocation. Knowing just where the femoral head is greatly helps the surgeon to make a prognosis of the result from a manipulative replacement. In wisely selected cases one may expect perfect results from replacing posterior dislocations. The percentage of perfect results from supracotyloid dislocations is less; and from anterior dislocations it is still less. I expect to get at least 70 percent of perfect hips as a result of my manipulative replacement. Denucè, in Bordeaux, France, had about 80 percent perfect results. This I believe was due to the fact that he did not begin to operate on these cases until about 1914, and because he was able to give better after-treatment than I have ever been able to give. Denucè's patients were treated in the hospital for many months—often for more than a year. My patients were kept in the hospital for only about three days. As soon as a child could be picked up from his bed without crying he was taken home, and only rarely did I see the patient again until the end of eight months; and in one instance the child was taken from the hospital two hours after the operation and carried home a distance of 200 miles. My unilateral cases walked by the end of two weeks, and often went to school. Denucè's patients did not walk at all until he believed the hip secure in the socket.

In selecting cases for operation, I prefer to have the child from 2½ to 3 years old, with a shortening of an inch to an inch and a quarter. But the age is of much less importance than the extent of the upward displacement. My oldest patient was a girl of 13 years with only an inch of shortening because of a mushroomed head and a short femoral neck. It relapsed. My next oldest was 11 years with an inch and three-quarters of shortening. The result was perfect, and the head still in after twenty years, although the socket showed shallow in the X-Ray film and the head was not quite normal in contour. In the early days I operated on one child of 16 months and one of 19 months. Both relapsed because the muscles were not shortened enough to hold the head in the socket. I want an inch of shortening, and not more than 2¼ inches. I have replaced cases with more, one with 3¼ inches, and the hip was left with very little movement. One bilateral case with 2¼ and 2½ inches at the end of eight years walked perfectly, but had difficulty in going up and down stairs. This case was a large girl of ten years, while a small child of six years, with 2⅛ and 2¼ inches upward displacement, had a perfect result. I have operated at the Children's Hospital and the Massachusetts General Hospital in Boston, in Providence, in New York City and Rochester, N. Y., in Philadelphia and Pittsburgh, Pa., in Cleveland and Cincinnati, O., in Detroit, Benton Harbor and Kalamazoo, Mich., in Fort

Wayne, Kokomo, Rushville and Evansville, Ind., in Chicago, Rockford and Rock Island, Ill., in Sheboygan, Fond du Lac and Madison, Wis., in Chattanooga, Tenn., in Dubuque, Des Moines and Sioux City, Iowa, in Lincoln, Nebr., in Sioux Falls, So. Dakota, in St. Paul and Minneapolis, Minn., in Denver, Col., in San Francisco, Calif., and in Albuquerque, N. M. Some years ago the American Orthopedic Association appointed a committee to investigate final results, and recommend a method of treatment. Of the cases reported on, 46.5% were my cases. The committee recommended only two methods: that of Ridlon and that of Denucè.

My treatment is as follows: The patient is fully anaesthetized. Ether is my choice. I operated once with the patient under Nitrous Oxide gas at the Lakeside Hospital in Cleveland, and have replaced a few hips when Etheline gas was used. The patient's hips are raised about four inches on a sand-bag; an assistant holds down the pelvis and thigh on the other side; for a left hip the crotch of the operator's left hand is put in the crotch of the patient, with the thumb in front of the socket and the first, second and third finger-tips on the femoral head, neck and greater trochanter of the dislocated hip which can be felt when the thigh is flexed; the bent knee is in the palm of the operator's right hand; the thigh is flexed until the head reaches the lower border of the socket (In a few older children with upward displacement of more than two inches it may be necessary to rotate the thigh inwards to bring the head low enough); then the thigh is abducted; presently the femoral head can no longer be felt by the tip of the index finger; and then presently it can be felt by the thumb in front; and then it goes into the socket—more or less into the socket. In only a few cases does the operator get the sensation of the cartilage of the head in contact with the cartilage of the socket. It is in most instances just looking into the socket through the narrowed capsule and cotyloid ring. In very many instances the bent knee can now be released, and the position does not change. But sometimes the thigh when released will adduct, and the head slips out. Then it should be again replaced, bored into the socket, and possibly the knee be further pressed backwards, even beyond the transverse plane of the body, until it no longer springs up and the hip goes out. The thigh is then in 90 deg. flexion, 90 deg. abduction, and rotated out 45 deg. In this position the greater trochanter lies below the head, and the head is in a position from which it may slip to the front of the socket. The back of the thigh looks downward, and its outer side backwards. If it is rotated inwards towards the normal position where the back of the thigh looks backwards, and its outer side outwards it is apparently in some cases more perfectly in the socket. But to retain it in that position—that is to say, without any rotation—the plaster splint must be carried below the flexed knee, in which position the child cannot walk on the foot of that leg. In all but a few cases I choose to risk the head slipping forward for the sake of having the child walk early, and much, to force the head deeper and deeper into the socket. When a dislocated hip has been replaced, the bent knee cannot be straightened because of the short ham-string muscles. But short ham-string muscles must not be accepted as a proof that the head is in the socket; it may be anywhere in the neighborhood of the socket, or even in the obdurator foramen. The operator must be able to feel the head in the socket. Then with the thigh flexed and abducted 90 deg. and rotated out 45 deg., the plaster cast is put on. The child is covered with stockenette that fits without a wrinkle; a muslin bandage is drawn through between the skin and the stockenette in front of both hips, for use as a scratcher; sheet-wadding bandages are wrapped around the thigh and body up to the ribs to the thickness of about half an inch; additional wadding is placed over all prominent bony points; and this is bandaged down firmly and smoothly with an ordinary roller bandage, to give a smooth surface to the inner side of the plaster splint. The plaster splint ought to be a very thick one—so thick that it will not bend or break even if wetted by urine occasionally. At the top it is trimmed down to just below the border of the ribs; above the unoperated thigh it is trimmed off enough to allow flexion of that thigh to the sitting posture; at the knee, off at the back, the outer and front sides to allow right-angled flexion of the knee, but on the inner side of the thigh it should be left to extend down below the inner condyle. The splint is then covered with stockenette. The child is put in bed with the buttocks and the thigh of the replaced hip raised, so that the child lies in the cast, and the weight of the cast not on the child. The child leaves the hospital as soon as it can be picked up without crying. The unilateral case should walk at the end of two weeks; the bilateral case should stand and begin to walk at the end of two months. From the beginning gentle straightening of the knee should be done, and the child encouraged to kick the knee straight. This forces the femoral head more deeply into the socket. Nothing else is done

until the end of eight months, when the cast is removed. Then the parents are requested to daily flex the thigh, carrying the bent knee towards the shoulder of the opposite side, and rotate the thigh inwards. It may require many months to bring the leg down into adduction beside the other leg. The longer this takes, the surer is the hip of staying in. The parents are told that if at anytime the leg comes down suddenly next the other leg, and no longer seems to be longer, the hip has probably slipped out, and the child should be brought for examination. If a hip slips out, nothing ought to be done until the upward displacement has again become an inch or more. Then it should again be replaced. Many years ago I replaced a hip in a little girl from Benton Harbor, Mich. It relapsed. At the end of a year at the urgent solicitation of the parents I replaced it again. Again it slipped out. Then I waited five years, and replaced it, and that time it stayed in.

The Denucè manipulation is substantially the same as mine, except that he carries the flexed knee to the opposite shoulder before abducting it in all cases. This I feel is not necessary. He does not mention that he places his hand in the child's crotch with thumb in front of the socket and fingers on the head, neck and greater trochanter of the femur in order to know just where the head is while it is being replaced. At the end of from two to four weeks the cast is bivalved, the front half lifted off, and the child carefully placed in a tank of brine, salt enough to float him. Here as he floats he is encouraged to move the thigh. Then he receives massage, and is exposed to sunlight. The child is not permitted to stand or attempt to walk until the surgeon from his clinical examination and from the X-ray film is confident that the hip is secure.

Putti, of Bologna, Italy, has operated upon a large number of cases. He prefers to operate on them as young as possible, even before they are "house-broke." Instead of a plaster cast he puts a wedge-shaped mattress between the child's abducted thighs; and the mattress is covered with water-proof material.

Osteotomy of the femoral shaft to correct real or imaginary bending of the femoral neck by rotating the fragments one on the other was done by Sherman of San Francisco and Hibbs of New York, and is now being done by Gaenslen, of Milwaukee, and others. Hibbs made the osteotomy before a bloodless replacement on his special table, and Sherman did his about two months after replacement by the open operation. These operations seem to me to be unnecessary and absurd. All are done with the intent to turn the femoral head backwards into the socket—the assumption being that all the necks are twisted forwards. Why these necks are twisted — if any are twisted — has not been explained. One might assume that when a femoral head is displaced forwards the neck might tend to bend in that direction during the period of growth. But if this is what happens it must be assumed that when the head is displaced backwards the neck tends to bend in that way; and when displaced upwards, tend to bend upwards; but these bendings are ignored by the osteotomists. Further, none of them have reported any considerable number of final results to compare with the results from non-osteotomized cases.

And the same may be said of the open replacements—the Hoffa operations and their modifications. Galloway, of Winnipeg, Canada, is now the only orthopedic surgeon I know who does the open operation in all cases. He has not only not reported his results, but when a former assistant of mine visited him to observe some of his results none were shown—all were too far away! The relapsed hip from manual replacement that Hoffa did for me in 1904, and which was in when the plaster splint was removed, relapsed by the end of six months. My single case relapsed. Bradford, of Boston, who had a large experience, manually replaced one hip of a bilateral case. It relapsed. He then replaced it by the open method, and again it relapsed. The other hip he replaced with the machine of Lawyer Bartlett and it remained in. I am willing to admit that an open operation designed to replace and anchylose the hip joint is worthy of consideration—if one prefers and anchylosed hip to a relapsed dislocation with good movement. Just now the so-called shelf operation is being done by several orthopedists. This was first done by Alexander Hugh Ferguson in Chicago in the early years of this century. It was a child of proper age for manual replacement, and with only about an inch of shortening. Ferguson did not attempt to replace the dislocated head, but just turned a flap of the outer table of the ilium down over the head. Six years later I examined the child and found the head just where he had put it, and the false joint had a good range of motion. But why do such an operation on a case that could have been readily replaced manually, and with a 70%, or better, prospect of a perfect joint?

THE RHODE ISLAND MEDICAL JOURNAL

Owned and Published by the Rhode Island Medical Society
Issued Monthly under the direction of the Publication Committee, 106 Francis Street

FREDERICK N. BROWN, M.D., *Editor*
309 Olney Street, Providence, R. I.

CREIGHTON W. SKELTON, M.D., *Business Manager*

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Advertising matter must be received by the 10th of the month preceding date of issue.

Advertising rates furnished upon application to the business manager, CREIGHTON W. SKELTON, M.D., 106 Francis St., Providence, R. I.

Reprints will be furnished at the following prices, providing a request for same is made at time proof is returned: 100, 4 pages without covers, $6.00; each additional 100, $1.00; 100, 8 pages, without covers, $7.50; each additional 100, $2.80; 100, with covers, $12.00; each additional 100, $4.80; 100, 16 pages, without covers, $10.50; each additional 100, $3.00; 100, with covers, $16.00; each additional 100, $5.50.

SUBSCRIPTION PRICE, $2.00 PER ANNUM, SINGLE COPIES, 25 CENTS.

Entered at Providence, R. I. Post Office as Second-class Matter.

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EDITORIALS

STOP THE LEAK!

The recent economic depression has wrought many social adjustments, some for good and some for evil. The more fortunate well-to-do people have of necessity developed a keener appreciation of "How the other half live" and have risen nobly to the occasion with unparalleled altruism and generosity creating and financing great hospitals and clinics. All of this is commendable and deserves the admiration of all, and especially the medical profession who perhaps have always been closest to the suffering and poverty-stricken of their respective communities.

But along with such provision to help the afflicted there has grown a great deal of abuse of so-called charity by persons who could well afford to pay at least a modicum for the benefits which they obtain, and which were in their original planning intended only for those in dire want.

Many of these beneficiaries were and are ignorant of the fact that they are literally stealing aid from those for whom it was intended. Others, as may readily be proved by those cognizant of the

facts, are deliberately misrepresenting their financial status and demanding free or nearly free care, under false pretenses.

And the Doctors are left "Holding the Bag."

As to the remedy for a situation that is assuming the proportions of a national disgrace,—there can be no relief until definite criteria of deservedness based on investigated facts, are established.

Social workers are neither qualified by inclination, training, or business acuity to determine who and who is not entitled to the benefits of free clinics. This has been repeatedly demonstrated and is the reason for the great proportions to which these abuses have progressed. There is need for a definite, trained financial agent or agency for each clinic large or small, under constant oversight of a staff committee in order to even approach a solution of the problem.

A simpler and more effective method is to receive no free or partly free patients except through certification from the Director of Public Aid. Such a rule in operation at a large hospital in one of our own cities has already demonstrated its necessity by the glaring reduction in the applications for admission to free clinics.

If we take a less than belligerent attitude and take it now, through organized effort, we shall see ourselves retired from private practice and engaged in a form of "State Medicine" but without remuneration in a very short while.

Already free clinics are taking hundreds of patients out of our offices who could and should reward the physician for his painstaking, effective and kindly care. It is time we got together and made constructive rules to stop the nefarious "Hold Up" of our profession.

Rhode Island Hospital Clinical-Pathologic Conference

Case No. 1, Presented by Dr. John B. Ferguson

M. S. Admitted March 3, 1932; age 68; occupation, housewife; married; white; female.

C. C.: Pain in upper right quadrant.

P. I.: Patient has been having pain for past year in upper right quadrant. Original onset was not very severe. About 3 months before admission pain returned and since that time has been getting progressively more severe.

P. H.: Mastoid operation, operation for tumor of cervix, no other hospitalizations, accidents or operations.

Progress Notes

3/7/32. Appendectomy and Cholecystectomy on the 4th. Good post-operative recovery.

3/18/32. Wound drained and a rubber tissue drain was inserted for 24 hours.

3/25/32. Still has indurated area to rt. of wound. Drainage increased some.

4/3/32. Wound draining less. Runs a persistently high temperature. Complains of pain in right ear. The drum is infected and slightly bulging. Phenal glycerine drops ordered.

4/6/32. Drum was opened two days ago, draining small amount of pus. Condition does not appear very good.

4/9/32. Temperature is lower today. Breathing, however, is difficult. Shows signs of beginning consolidation at the left base, probably a hypostatic thing.

4/11/32. Condition took a decided turn for the worse during the night. Both lungs filling rapidly. Prognosis very bad.

4/12/32. Condition became progressively worse and she expired at 1:30 A. M. Permission for post-mortem examination granted.

X-Ray, April 9, 1932: Examination of the mastoids shows both mastoids to be of average size and made up of pneumatic cells. The right mastoid shows no definite evidence of pathology. The left mastoid is dull and is consistent with mastoiditis.

3/4/32. Operation: Appendectomy and Cholecystectomy — Usual upper right rectus incision. Gall bladder found greatly enlarged, distended. No stones felt. Liver adherent to ant. wall with old adhesions. Not disturbed. Stomach and duodenum normal. Pancreas greatly enlarged, hard, not particularly irregular. Gall bladder removed from below upwards, isolating with ligating duct first. Bladder closed over with running silk catgut stitch. One cigarette drain to gall bladder fossa. Short, stubby appendix dissected from wall of cecum, plastered down by old adhesions. Wound closed in usual layers.

Laboratory Data

Urine: S. Yellow, acid, Sp. Gr. 1022, sugar negative. Least possible trace albumen. Slight amount of sediment.

Urine: Amber, Acid, Sp. Gr. 1020, sugar negative, albumen negative.

Dr. Ferguson: The history on this case is rather meagre. I first saw this woman in January, 1932. At that time she came in with a history of having

had gas and indigestion for the past ten years, etc. (Reads mimeographed copy.)

There was nothing in the extremities except marked thickening of the joints from hypertrophic arthritis. She had had an operation of the cervix. She had never been jaundiced. We sent her over for some X-Ray pictures of the gall bladder on January 27. The report reads: "Examination of the gall bladder after oral administration of dye shows the gall bladder somewhat larger than usually seen. The gall bladder is well outlined with the dye. It did not empty after the usual meal. Films were again repeated in six hours and the gall bladder shadow shows good concentration of the dyes. Findings are consistent with a poorly functioning gall bladder. There is no evidence of opaque calculi."

On the 4th of March we operated. The usual right upper rectus incision. Gall bladder found greatly enlarged, distended. No stones felt. Liver adherent to the anterior wall with old adhesions. Not disturbed. Stomach and duodenum normal. Pancreas greatly enlarged, hard, not particularly irregular. (We thought it might be a chronic pancreatitis.) Gall bladder removed from below upwards, isolating with ligating duct first. Bladder closed over with running silk catgut stitch. One cigarette drain to gall bladder fossa. Short, stubby appendix dissected from wall of cecum, plastered down by old adhesions. Wound closed in usual layers. She went along with a normal temperature until the 16th of March. On that day she got a little temperature and I looked at the wound. The dilated sinus had evacuated pus. From that time on she drained a good deal of pus and on the 27th of March she had taken cold and said she had a pain in her right ear.

On the 3rd of April the ear showed that the right drum was infected and slightly ruptured and the ear man came over and opened the drum and got quite a little pus and ordered some A. B. irrigation. In a day or two she complained of pain in the left ear. That showed some trouble there and it opened itself the next day. The temperature came down gradually but never satisfactorily and on the 9th of April she showed signs of consolidation at the left base. At the same time the picture of the mastoids showed both to be of average size and made up of pneumatic cells. The right mastoid shows no definite evidence of pathology. The left mastoid is dull and consistent with mastoiditis.

On the 11th of April she became very ill and both lungs filled up rapidly. The chest became very solid and she became progressively worse and died at 1:30 A. M.

Permission for post-mortem was granted.

DR. CLARKE: The only X-Ray pictures we could find were the ones of the mastoid. I presume the others were taken on the outside.

DR. FERGUSON: They were taken here.

Discussion

DR. COOKE: This case came to me and I had been puzzled over it for twenty-four hours. From the history and the X-Ray examination it is clear that she had trouble with her gall bladder. On the other hand, one feels that possibly the gall bladder was not the cause of the trouble. In the first place she had a tumor of the cervix removed. I don't know whether that was benign or malignant. At the operation the woman presented an enlarged pancreas and an enlarged gall bladder, yet there was no jaundice. If she had cancer at the head of the pancreas, the jaundice would be painless. At operation a large gall bladder was found and removed. There were no stones. The liver showed a good many adhesions about it. The possibility there is of an old inflammatory process. To my mind, there is a possibility of some malignant condition there and to my mind it is possible to have a tumor of the pancreas which does not involve the head of the pancreas. This woman went along about ten days and then developed a temperature, following which she had an infection of both middle ears. X-Ray shows involvement of the mastoid on the left. I cannot say if that was in connection with pneumonia or whether it was a new growth. Certainly, going back to the history of ten or twelve years, it is very suggestive of gall stones, and yet this woman did not have gall stones. I have no doubt that this gall bladder was pathological under the microscope. A good many gall bladders have been removed (chronic cholecystitis) where the symptoms have not been relieved and where gall stones were not found. What more I know about the case is what Dr. Ferguson said, and knowing nothing about the post-mortem I should think the primary lesion is in the pancreas. I am sorry I cannot be positive but that is what I would imagine.

DR. DEWOLF: What was the previous history of the woman's condition for the past ten or five

years? You go back one year and that is all. Did she lose weight?

DR. FERGUSON: That history is not correct. She had been ailing for ten or twelve years with typical gall bladder symptoms, but she had not lost much weight.

Demonstration of Post-Mortem Material

DR. CLARKE: I have no gross specimens on this case, so I will have to tell you about the post-mortem findings rather than show the actual specimens. The first thing noted at the post-mortem was in doing the external examination. Dr. Newel did this post-mortem and he describes a "partially healed upper right quadrant surgical incision with a small fistula opening in the lower part of the wound. This fistula is probed and found to extend up and down beneath the peritoneum, but does not lead to the gall bladder bed but is traced down around the caecum, where it is lost in numerous adhesions." After he got into the peritoneal cavity, there were numerous adhesions between the diaphragm and the liver and also numerous ones around the gall bladder area and in the region of the caecum. The stump of the appendix was inverted and just beyond it there is found a small "perforation from the caecum out into a cavity formed by adhesions which leads to the sinus tract previously described. In this cavity is found a small piece of suture material."

The next thing is the lungs. The right weighed 1000 grams and the entire upper right lobe was solid. The lower lobe of the right lung was also solid and there was a mixture of red and gray — so there was a lobar pneumonia involving both the upper and lower lobes of the right lung.

The spleen was a typical pneumonia spleen.

The pancreas consists of a gray, granular, hard mass measuring 6 x 8 cm. On section no pancreatic tissue is recognized. The liver weighed 1400 grams. It was studded with gray, granular nodules, the largest of which measures 1.8 cm. in diameter. There was nothing in the genitalia. The cervix was distorted, possibly the result of some previous operation, but there is no tumor in the cervix or uterus at post-mortem.

So there were three distinct lesions:

Carcinoma of the pancreas, involving not only the head of the pancreas but it had replaced the pancreas.

There were metastatic lesions from this in the liver and, in addition, there was this perforation of the gut, retroperitoneally and draining at the operative wound, and in addition there was a terminal lobar pneumonia.

(*Slides shown*)

Q.: Was there any metastasis in the lungs?

A.: The only metastasis found were in the liver.

DR. CHASE: That thing around the appendix—where did you say it was?

DR. CLARKE: It is in the caecum about 1 cm. from the stump of the appendix.

CASE REPORTS

CASE REPORTED BY DR. MCCAFFREY

December 8, 1931

It seemed to us that the diagnosis in this case pointed to something in the cortex or something in the meninges. In considering the meninges, we thought first of the possibility of a subarachnoid hemorrhage, but this was ruled out by the absence of blood in the spinal fluid. Other forms of cerebral hemorrhage were ruled out by the general spasticity of the extremities. Abscess of the brain was considered. There seemed to be no primary focus to which we could attribute an abscess. Syphilis was considered, but this seemed to be ruled out by the negative serology. Tuberculosis with meningitis was a possibility which, I think, in this case should be considered. However, the long duration of the sickness is probably against tuberculous meningitis. Most cases live only four weeks. Then again against tuberculous meningitis is the finding of 121 mgs. of sugar in the spinal fluid. That is too high for tuberculous meningitis. No history of cough. No cough while patient was in the hospital. We also considered Paget's disease of the bone, because the patient had a rather large head and these areas of calcification which were seen in the X-Ray plate did in some way suggest the possibility of Paget's disease of the bone. Before we had a chance to look further the patient expired, but we did have post-mortem X-Ray examination of the pelvis which showed nothing in the pelvis to suggest Paget's disease of the bone. It seemed to us that the X-Ray of the lungs probably solved the difficulty. This increased density in the right upper lung was in an area where we found dullness with bronchial breathing. It seemed to us that the patient probably had a new growth primary in the lung with metastasis to the brain. There was nothing in the physical examination to indicate the location of the cerebral tumor unless we considered the speech

defect, in which we might look for a growth in the left hemisphere. There was not anything to help us except the occipital headaches."

Demonstration of X-Ray Films

DR. BATCHELDER: The first films which were taken are those of the skull. There is a rather faint shadow right here. The report should have read: "Lateral views of the skull showed two areas of calcification two inches above the sella turcica." Those show in both the right and left lateral views. It is brought out a little more clearly here. The shadows are of much the same appearance, except one is larger than the other. There is a fairly well defined border to the smaller one. Rather indistinct on the larger. A more or less similar relation holds on the other side. It is not the typical appearance of calcification in the choroid, but in view of there being two we thought that that was probably the explanation. There are no signs indicating increased introcranial pressure. Film of the chest shows the result of considerable motion during the exposure, but it does show this large mass in the upper right lung filling over about the same density of the upper mediastinal shadow. There is also a good representation of the heart shadow which does not appear enlarged. Of course, many fine changes in the lung fields would be obliterated by the motion. In the report both the possibility of a new growth and aneurysm were suggested. From the location it could very well be an aneurysm, but a point I think I mentioned before in these meetings that I find usually holds true is that there is usually an enlargement of the heart shadow with an aneurysm. I understand that it is not one hundred percent true but it is true in a large percentage of cases. Another possibility which might be mentioned would be collection of glands from a Hodgkin's disease. In that condition, however, the shadow usually has a lobulated border, quite definitely so, as the mass is made up of a collection of enlarged glands. So from the X-Ray we come to the conclusion that it was most likely a malignancy arising in the bronchus or in the trachea.

The films taken at post-mortem of the pelvis are perfectly normal. A spot film of the lateral skull shows these shadows more clearly than the other. Again these shadows cannot be positively identified. There is a suggestion of a very faint shadow that appears to the left of the mid-line.

DR. HUGHES: As to the possibility of localization, probably the enlargement of the left pupil points to involvement on that side. We have a difference in the jugular pressure. Whether it is definitely on the left side or not, the headache being more toward the occiput would tend to suggest that the process might be more on the left side. The chest X-Ray picture as interpreted seems to indicate malignancy. The cell count and colloidal gold and the calcification in the brain could be due to a tumor of some sort. Metastatic tumors in the head often give the first clinical signs of tumors in the chest or tumors in the prostate. A doctor sometimes may not know there is any tumor in the prostate or chest until he finds something in the head.

Demonstration of Post-Mortem Material

DR. CLARKE: Here is the apex of the left lung. At this point is a large bronchus which is plugged by gray, granular, pliable tumor tissue. When we cut across it here is this large tumor mass infiltrating the lung tissue. The histology of that tissue is typical of epidermoid carcinoma originating from the bronchi.

Back here in the right occipital lobe growing out of the cerebrum is this large tumor mass, largely necrotic. The histology is the same as that of the lung tumor, so it is primary carcinoma of the bronchus with metastasis to the brain. I have both of the chorioid plexuses here. There is not much to see grossly, but when you cut it with the knife there is a little calcification in each chorioid plexus.

DR. MCCAFFREY: This case was presented to bring out the fact that chronic disease primary elsewhere in the body frequently manifests itself by neurological signs. I have in mind two other cases that first presented themselves by neurological signs. One was a case of Paget's disease of the bones which first showed the signs of meningitis. This patient was taken sick with a fever. Spinal fluid examination revealed increased pressure with massive coagulation of the fluid. X-Ray of the head and the pelvis showed the typical changes of Paget's disease. In this particular case there had been nothing of which this patient complained until he was taken sick and developed the signs of meningitis. In this particular case we felt that the meningeal symptoms were probably due to an irritation of the spinal cord due to the heavy weight of the head. The head increased in size. The other case which first presented neurological signs was a case which we had recently of tuberculous meningitis. This boy had had pneumonia last winter and had not made a satisfactory convalescence. He did

not regain his normal weight and complained of nothing except general weakness and loss of appetite. He was suddenly taken sick with fever. He lost interest in his recovery. Examination of his lungs, which were not at all suggestive of a cough or chest condition, revealed a tuberculous process, and spinal fluid was consistent with tuberculous meningitis. This case was presented to bring out the point that chronic cases frequently first manifest themselves neurologically.

SOCIETIES

RHODE ISLAND MEDICAL SOCIETY

REPORTS OF COMMITTEES

(Continued from the August issue)

COMMITTEE ON NECROLOGY

During the past year, the Rhode Island Medical Society has lost through death seven active members, one honorary member and one non-resident member.

Dr. Frederick T. Rogers died on August 23, 1932 in his seventy-fourth year. Dr. Rogers was born in New York State in 1859. He graduated from the University of New York Medical School in 1882. He later did post-graduate study in the Manhattan Eye and Ear Infirmary. He was surgeon to the Eye Department of the Rhode Island Hospital for many years and consultant to St. Joseph's Hospital, and Charles V. Chapin Hospital, Pawtucket Memorial Hospital and the Woonsocket Hospital. He was President of the Providence Medical Association from 1901-1902 and Treasurer of the Rhode Island Medical Society from 1897-1902. He was President of the State Society in 1911-1912. He was Vice-Chairman of the Ophthalmological section of the American Medical Association in 1911, and President of the New England Ophthalmological Society in 1912. He was a member of many other medical societies.

Dr. Richard F. Duncan died on December 9, 1932, in his sixty-eighth year. Dr. Duncan was born in Massachusetts in 1865, and graduated from the Albany Medical College in 1889. About twenty years ago he did post-graduate work in Ophthalmology at the Harvard Medical School. He was a member of the staff of the Eye Department at the Rhode Island Hospital, St. Joseph's Hospital and Sayles Memorial Hospital.

Dr. Cornelius J. Mahoney died on January 3, 1933, at the age of fifty-eight. Dr. Mahoney was born in East Providence in 1875. He graduated from the Harvard Medical School in 1898. He was a member of the staff of St. Joseph's Hospital for many years.

Dr. George R. Barden died on January 29, 1933, at the age of sixty-five. Dr. Barden was born in Scituate, R. I., in 1867 and graduated from the Bellevue Medical College in 1896. In 1902 he went to the Columbus Hospital in New York City, where he was an interne for three years. During this period he completed a course at the New York Post-Graduate Hospital. In 1916 he was made a Fellow of the American College of Surgeons. He served as surgeon and consulting surgeon to the St. Joseph Hospital and the Rhode Island Institutions.

Dr. Gilbert Houston, 2nd, died on February 21, 1933, at the age of thirty-nine. Dr. Houston was born in Rhode Island in 1894. He graduated from the University of Vermont, College of Medicine, in 1918. He had practiced in Pawtuxet Valley since 1919. Dr. Houston served in the Medical Out-Patient Department of the Rhode Island Hospital and Pawtucket Memorial Hospital. He was, at one time, President of the Kent County Medical Society.

Dr. Henry A. Cooke died on March 17, 1933, at the age of sixty-seven. Dr. Cooke was born in Groton, Mass., in 1866. He graduated from Harvard Medical School in 1896 and was an interne at the Massachusetts General Hospital for two years thereafter. He began private practice in Providence in 1899, and his long career as a prominent practitioner was interrupted in 1906 by two years devoted to regaining his health at Saranac. He returned to Providence and continued in active work until three years ago. Dr. Cooke was visiting physician for the Rhode Island Hospital for many years.

Dr. Raynor Woodhead died on April 11, 1933, at the age of fifty-seven. Dr. Woodhead was born in 1867 and graduated from Tufts Medical School in 1898. He practiced medicine in Valley Falls, R. I., for over thirty years.

Dr. William W. Keen, a world renowned surgeon and an honorary member of the Rhode Island Medical Society, died in Philadelphia, on June 7, 1932.

Dr. Winthrop A. Risk, a non-resident member, died in Washington, D. C., on May 2, 1933, at the age of sixty-four. Dr. Risk was born in Massachusetts in 1869 and graduated from the Harvard Medical School in 1893. He became superintendent of the Rhode Island State Home and School on June 1, 1905, and filled that position for twelve years and two months. For the past ten years he had been resident physician at the Tuberculosis Hospital at Washington, D. C. He served on the staff of the Gynecological Department of the Rhode Island Hospital. During the World War he served as a Captain in the Medical Corps of the United States Army, and from 1919-1929 he was a Major in the Medical Reserve Corps. He was Treasurer of the Rhode Island Medical Society for ten years and held the same office in the Providence Medical Association for nineteen years. He was business manager of the RHODE ISLAND MEDICAL JOURNAL for four years.

HERMAN A. LAWSON, *Chairman.*